UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY
CAMDEN VICINAGE

IN RE: VALSARTAN, LOSARTAN, AND IRBESARTAN PRODUCTS LIABILITY LITIGATION **This Document Relates to All Actions**	MDL No. 2875 Honorable Robert B. Kugler, District Court Judge Oral Argument Requested

DEFENDANTS' REPLY IN SUPPORT OF THEIR JOINT MOTION TO EXCLUDE THE OPINIONS OF STEPHEN LAGANA, M.D.

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When the rhetoric is peeled back, Plaintiffs' Opposition ("Opp.") amounts to an argument that Dr. Lagana correctly interpreted the studies he chose to read. Rule 702 and *Daubert* require more. Plaintiffs have not and cannot show that Dr. Lagana considered all relevant studies rather than selecting only those that supported his conclusion-driven analysis. Plaintiffs also have not refuted Defendants' showing that Dr. Lagana's opinions fail to assist the jury with interpreting the studies he selected in a way that is relevant to the general causation inquiry.

As Defendants' Motion made clear, Dr. Lagana's opinions are not methodologically sound and, as a result, cannot assist a jury. The fatal flaw in Dr. Lagana's methodology is that he *presumed* exposure to a carcinogen causes cancer. Plaintiffs' dismiss that statement as "an isolated introductory background statement," but Dr. Lagana's report repeatedly doubles down on that flawed premise. Compounding the problem, he ignored or minimized key studies that contradicted his conclusion in further deviation from acceptable scientific methodology.

Further, Dr. Lagana made clear that his opinions do not address general causation. He explicitly lays out his methodology: he is considering "the potential etiologic role of a specific carcinogen," ignoring the key question in this litigation — whether the levels of NDMA and/or NDEA in some valsartan containing medications cause cancer in humans. Stephen Lagana Report (the "Report"), at 12.

Dr. Lagana's premise and conclusions are consistent with his lack of relevant expertise: Dr. Lagana is not an epidemiologist, toxicologist or cancer researcher; he is an anatomical pathologist, focused on evaluating tissue specimens from patients suspected of having cancer. That expertise does not qualify him to opine on whether substances are carcinogenic in the first place, much less to opine whether potential carcinogens are actually carcinogenic in humans at certain doses. Dr. Lagana's recitation of select epidemiology studies provides no meaningful assistance.

Plaintiffs' Opposition does not credibly refute any of these points. Instead, Plaintiffs expend pages mischaracterizing the testimony of Defendants' experts and obfuscating Dr. Lagana's methodological shortcomings.[1] Accordingly, Dr. Lagana's opinions should be excluded.

ARGUMENT

I. DR. LAGANA'S METHODOLOGY IS FLAWED AND UNRELIABLE.

A. Dr. Lagana Confirmed at His Deposition and in His Report That He Did Not Start With a Null Hypothesis.

The question in this case is whether NDMA/NDEA are human carcinogens at the levels detected in valsartan containing drugs. Accordingly, it is Plaintiffs' burden to present a preponderance of evidence that NDMA/NDEA can cause cancer in

[1] For example, contrary to Plaintiffs' assertions, Defendants' experts do not "concede" NDEA and NDMA are probable human carcinogens. They testified that IARC has classified them as such, but also have stressed the importance of dose in specific analyses – a key factor Dr. Lagana wholly ignores.

humans at the dose, duration, and route of exposure relevant in this case. Dr. Lagana's opinions do not aid Plaintiffs in sustaining that burden.

Courts recognize that such an opinion must be rooted in epidemiology and *must start from a premise [i.e., the null hypothesis] that assumes the exposure did not cause the observed effect (cancer)*: "the scientific methodology that allows testing of the hypothesis that Substance A causes Effect B." *Soldo v. Sandoz Pharms. Corp*., 244 F. Supp. 2d 434, 533 (W.D. Pa. 2003); Reference Manual on Scientific Evid., Third. Ed. (2011) at p. 315 (defining hypothesis test: "the idea is to see whether the data conform to the predictions of the null hypothesis").

Dr. Lagana's opinion, by contrast, is 'rooted' in his experience as an anatomic pathologist and improperly presumes that the NDMA exposure caused the cancer. Plaintiffs cannot escape Dr. Lagana's own statement in his report:

> "…for any patient who develops cancer and is known to have a significant exposure to a probable human carcinogen . . . **it should be assumed** that the carcinogenic exposure at least increased the risk or contributed to the subsequent cancer, unless there is a convincing body of evidence to suggest that the carcinogenic insult is null with respect to the specific cancer in question." (Rep. at 11 (emphasis added)).

Plaintiffs mischaracterize this as an "isolated introductory background statement from Dr. Lagana's report regarding the real-world application of a differential diagnosis to determining the cause of an individual's cancer." Opp. at 7. In fact, the statement is anything but isolated — the report states, "The crucial point

is that ***we start from an assumption that exposure to a human carcinogen contributed to carcinogenesis*** in a patient with cancer"; Dr. Lagana then searched for "convincing evidence to the contrary." Report, at 11 (emphasis added). In other words, he explicitly assumes the affirmative hypothesis (not the null) and looked to see whether it can be *disproven*. That is exactly backwards. Plaintiffs attempt to minimize the impact of this flawed methodology by arguing Dr. Lagana undertook an unbiased review of the evidence, quoting his statements that he formed an opinion after review of literature. But, because his opinions were guided by a flawed premise, they reverse Plaintiffs' burden of proof and are unscientific and unreliable.

Dr. Lagana's flawed methodological approach is significant because it ignores the critical issues of dose, duration, and route of exposure, instead presuming that mere exposure to NDMA in valsartan is sufficient to have caused plaintiffs' cancers. But, it is a scientific fact that NDMA is ubiquitous in the environment and exposure occurs every day from multiple sources. *See* Ex. A, at p. 6.[2] Many studies ignored by Dr. Lagana in his analysis confirm that humans are exposed to NDMA daily, through a variety of means and routes of exposure, including to NDMA formed endogenously (in our bodies). *See infra,* Section 1(c). Thus, it is critical that any scientific opinion on general causation in this case evaluate dose, duration, and route

[2] A true and correct copy of et al., "Concise International Chemical Assessment Document 38: N-nitrosodimethylamine" is attached to the accompanying Affidavit of Seth Goldberg as **Exhibit A**.

of exposure, to determine whether there is any evidence NDMA and/or NDEA in valsartan are capable of causing plaintiffs' cancers. Dr. Lagana does none of this because he begins with an assumption of carcinogenicity. Dr. Lagana started with his conclusion, then worked backwards to find the supporting studies. The fact that Dr. Lagana's research methodology includes review of peer reviewed medical research does not cure his failure to consider all evidence or give appropriate weight to the evidence he did consider.

B. Dr. Lagana Failed to Give Any Weight to Critical Evidence and Plaintiffs Fail to Explain That Methodological Shortcoming.

Dr. Lagana failed to give weight to critical evidence that is directly relevant to the general causation inquiry. Most significantly, Dr. Lagana minimized the human epidemiology studies of patients exposed to NDMA-containing valsartan (Gomm and Pottegard), which address the very question at issue. His report, and Plaintiffs' Opposition, attempt to gloss over those studies' conclusions finding no statistically significant increased risk of cancer in valsartan users (except a nominally significant increased risk of liver cancer, in Gomm). Plaintiffs' only response is that Dr. Lagana considered both studies, noting that he cited them in his report and pointed out certain limitations of their findings. Opp. at 19-20. Plaintiffs fail to explain, however, why Dr. Lagana chose not to ascribe any scientific weight to the most directly analogous evidence available or why he preferred inapposite dietary studies.

Plaintiffs also attempt to buttress Dr. Lagana's opinion by asserting he evaluated an additional "valsartan epidemiology study that was ignored by some defense experts completely." Opp. at 20. The cited study, *Abrupt Increase in Reporting of Neoplasms Associated with Valsartan After Medication Recall*, is not an epidemiological study at all.[3] Opp. at 20-21. Rather, it's a review of the Federal Adverse Event Reporting System ("FAERS") data, which does not aid in answering the general causation inquiry at issue. Indeed, the FDA specifically instructs that FAERS data cannot be used to establish causation. *See* Ex. C at 2-4.[4] Pointing to this study as an additional "valsartan epidemiology study" is misleading and does nothing to bolster Dr. Lagana's opinions.

Similarly, Dr. Lagana ignores evidence that the level of endogenously-produced NDMA is orders of magnitude higher than exposure from any exogenous source. Dr. Lagana and Plaintiffs' Opposition attempt to minimize the obvious impact of daily exposure to endogenous NDMA by claiming there is "no solid evidence" of the *amount* of endogenous exposure to NDMA in our bodies. That is untrue and ignores that our bodies produce NDMA in excess of the FDA's

[3] A copy Al-Kindi, et al., *Abrupt Increase in Reportiong of Neoplasms Associated with Valsartan After Medication Recall,* is attached to the accompanying Affidavit of Seth Goldberg as **Exhibit B**.

[4] A copy of "Questions and Answers on FDA's Adverse Event Reporting System (FAERS)" *available at* https://www.fda.gov/drugs/surveillance/questions-and-answers-fdas-adverse-event-reporting-system-faers is attached to the accompanying Affidavit of Seth Goldberg as **Exhibit C**.

acceptable daily intake. There is ample evidence regarding the substantial levels of our endogenous exposure to NDMA.

One study estimated that endogenously formed NDMA account for between 45% and 75% of total exposure in humans. *See* Ex. D (Jakszyn, et al.).[5] Another study undertook an extensive review of the multiple available methods for estimating endogenous NDMA production and concluded that, "[d]espite the multiple possible sources of error in the estimates of endogenous formation of NDMA we have derived, it seems clear that formation rates approaching 1 mg/day are usual and in some individuals such formation can be significantly greater." *See* Ex. E (Hrudey, et al.) at 2197.[6] Instead of confronting the endogenous estimates, Dr. Lagana points to a study by Choi that does not address levels of endogenous production at all, focusing exclusively on whether dietary NDMA consumption can be linked to gastric cancer. *See* Ex. F (Choi, et al.).[7]

Plainly, Dr. Lagana's consideration of the dietary studies does not establish

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[5] A copy of Jakszyn, et al., *Endogenous versus exogenous exposure to N-nitroso compounds and gastric cancer risk in the European Prospective Investigation into Cancer and Nutrition (EPIC-EURGAST) study,* is attached to Defendants' Motion as **Exhibit D**.

[6] A copy of Hrudey, et al., *Drinking Water as a Proportion of Total Human Exposure to Volatile N-Nitrosamines*, is attached to the accompanying Affidavit of Seth Goldberg as **Exhibit E**.

[7] A copy of Choi, et al., *Consumption of precursors of N-nitroso compounds and human gastric cancer,* is attached to the accompanying Affidavit of Seth Goldberg as **Exhibit F**.

that he considered the role of dose and duration on carcinogenesis. His failure to consider the role of endogenous formation only compounds that shortcoming. Plaintiffs' only response is citation to inapposite case law. Opp. at 13. The *Marsee* case involved tobacco-specific nitrosamines, which are already known to be highly carcinogenic. *Marsee v. United States Tobacco Co.*, 639 F. Supp. 466, 469-70 (W.D. Okla. 1986) (finding animal and dietary studies valuable when dealing with tobacco, an established carcinogen). In short, Dr. Lagana fails to confront evidence that the human body produces between four and fifty times as much NDMA as was detected in the *highest* FDA measurement in any recalled valsartan tablet.

Compounding his failure to give weight to relevant evidence, Dr. Lagana does not thoroughly evaluate the evidence on which he does rely. Specifically, Dr. Lagana relies heavily on an occupational *inhalation* exposure study of rubber workers (Hidajat) but fails to present any analysis showing how oral ingestion and inhalation of the same compound are equivalent. Plaintiffs attempt to explain away this glaring deficiency by pointing out that the metabolic pathways are the same (Opp. at 24), but that is not the relevant inquiry. Rather, it is whether the levels of exposure in Hidajat are comparable to valsartan and whether the disparate exposure routes reach the same tissues and produce the same effects. Plaintiffs cite no evidence or study making this connection other than Dr. Lagana's *ipse dixit*. At bottom, Dr. Lagana

relies heavily on an inapposite exposure route to support his conclusions for numerous cancers, without providing sufficient basis in science or medical literature.

Similarly, and contrary to Plaintiffs' assertions, Dr. Lagana did not offer any evidence at his deposition to support his "inference" that individuals pre-disposed to cancer are at a higher risk from NDMA exposure. He admitted "there have not been any studies to date about patients with Lynch Sydrome exposed to NDMA and NDEA" and that this statement was his supposition (Lagana Dep. at 367:24-368:20).

Taken together, each of these shortcomings demonstrates Dr. Lagana's failure to consider relevant evidence, much less give it weight. He relies on a limited collection of studies which he asserts support his pre-formed conclusions. He then makes a number of unsupported inferences and assumptions to wave away contradictory evidence. His opinions are methodologically unsound and unreliable.

C. Dr. Lagana Failed to Consider Dose and Duration in His Analysis.

Dr. Lagana unequivocally testified that "there is no safe dosage and [] any exposure is likely to increase the risk of cancer." (Lagana Dep., Dkt. 1718-5, at 41:1-7.) Plaintiffs argue that Dr. Lagana's opinions "do take dose into account." Opp. at 22. Simply stating it does not make it so. Indeed, Dr. Lagana's failure to consider the role of endogenous NDMA is just one example. He also fails to provide any analysis of the minimum carcinogenic doses of NDMA and/or NDEA observed in animal studies.

As a preliminary matter, Dr. Lagana's blanket opinion that there is no safe dose of NDMA is directly contrary to the law on questions of general causation. *See, e.g., McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1242 (5th Cir. 2005) (citing Science for Judges I: Papers on Toxicology and Epidemiology, 12 J. L. & Pol'y. 1 (2003) ("***Dose is the single most important factor to consider*** in evaluating whether an alleged exposure caused a specific adverse effect. Often low dose exposures -- even for many years – will have no consequence at all, since the body is often able to completely detoxify low doses before they do any damage. Furthermore, for most types of dose-response relationships following chronic (repeated) exposure, thresholds exist, such that there is some dose below which even repeated, long-term exposure would not cause an effect in any individual") (emphasis supplied); *In re Abilify (Aripiprazole) Prods. Liab. Litig*., 299 F. Supp. 3d 1291, 1307-08 (N.D. Fl. 2018) ("[F]or the vast majority of substances, there are threshold doses below which no individual will respond… Consequently, a reliable expert opinion on general causation should address what levels of exposure to a drug increase the risk of adverse effects. Indeed, the expert who avoids or neglects this principle of toxic torts without justification casts suspicion on the reliability of his methodology"); *In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166,

(N.D. Cal. 2007) (excluding expert opinions that medication could cause claimed harm at lower doses than studied).[8]

By contrast, numerous experts for Defendants addressed the minimum carcinogenic dose shown to induce cancer in lab animals and demonstrated that those doses are orders of magnitude higher than the levels detected in valsartan. (*See, e.g.*, Dr. Chodosh Rep., Dkt. 1790-8, at 26). By contrast, Dr. Lagana does not even reference the amounts of each compound tested in the Peto study or the human primate studies. Plaintiffs' failure to provide any similar analysis connecting the actual exposure level of NDMA or NDEA in the affected valsartan to any study finding a carcinogenic risk in humans highlights their overall strategy to ignore dose and duration, treating all nitrosamines and all levels of exposure as equivalent. That approach is inconsistent with the law and scientifically unreliable.

Dr. Lagana also treats NDMA and NDEA interchangeably throughout his report, without evaluating them separately for carcinogenicity or any connection to the specific types of cancer reported. He simply assumes any cancer can be caused by either NDMA or NDEA and ignores the findings of the individual studies on

[8] At a minimum, this Court should exclude any opinion premised on a theory that there is "no safe dose" of NDMA and/or NDEA or that even one molecule of those chemicals could be carcinogenic. For further discussion, see Defendants' Joint Motion to Exclude Opinions of Dipak Panigrahy, M.D. *See* Dkt. 1716-1.

these compounds which say otherwise. *See* Ex. G-J.[9] For example, NDEA has been shown to cause liver tumors in non-human primates, while NDMA has not. *See* Ex. I at 143. Dr. Lagana similarly ignores the biological mechanisms and distinct enzymes responsible for metabolizing NDMA and NDEA, which are not present in all tissues. *See* Ex. G. Again, Dr. Lagana's expertise in pathology does not permit him to provide relevant scientific support for an opinion that NDMA and/or NDEA could cause cancer in any particular tissue, let along all the tissues in which he claims they are carcinogens. Dr. Lagana simply assumes that they can be treated identically. As a result, his conclusions and opinions are unreliable.

II. PLAINTIFFS FAIL TO SHOW DR. LAGANA IS QUALIFIED TO OPINE ON GENERAL CAUSATION OR THAT HIS EXPERTISE IS HELPFUL TO THE GENERAL CAUSATION ISSUE.

A. Dr. Lagana Is Not Qualified to Offer General Causation Opinions on Carcinogenicity.

Plaintiffs have failed to meet their burden of demonstrating that Dr. Lagana is qualified to render an opinion on general causation. The expertise Plaintiffs proffer from Dr. Lagana's background does not establish he has "extensive qualifications"

[9] A true and correct copy of Aiub, et al., "N-nitrosodiethylamine genotoxicity evaluation: a cytochrome P450 induction study in rat hepatocytes," is attached to the accompanying Affidavit of Seth Goldberg as **Exhibit G**; Magee, et al., "The production of malignant primary hepatic tumours in the rat by feeding dimethylnitrosamine" is attached as **Exhibit H**; Adamson, et al., "Chemical Carcinogenesis Studies in Nonhuman Primates" is attached as **Exhibit I**; and Thorgeirsson, et al., "Tumor incidence in a chemical carcinogenesis study of nonhuman primates" is attached as **Exhibit J**.

to offer the opinions he has given here. Opp. at 9.

Plaintiffs' point to Dr. Lagana's testimony that he "learned epidemiology in medical school," is "aware of epidemiological concepts," and has "worked with and published with epidemiologists." Lagana Dep., Dkt. 1718-5), at 122:10-19. But his study of epidemiology in medical school and his work with epidemiologists does not render him an expert in epidemiology, much less in carcinogenesis. *See* Opp. at 10. While medical doctors do not need to be epidemiologists in order to testify regarding epidemiological studies, they are still limited to testifying to areas within their expertise. *Calhoun v. Yamaha Motor Corp., U.S.A.*, 350 F.3d 316, 322 (3d Cir. 2003). Similarly, the fact that Dr. Lagana has co-authored articles with epidemiologists is not proof of his qualification to analyze epidemiology studies and offer general causation opinions based on those articles—just that he has worked with others who hold such qualifications.

Plaintiffs attempt to enhance Dr. Lagana's qualifications by stating he "takes general causation into account both in his clinical [sic] and his research and peer reviewed publications." Opp. at 4. That is a generous characterization and unsupportable since Dr. Lagana himself testified he does not render either diagnoses or causation opinions in the cancer patients whose tissue he examines. Dr. Lagana testified that he has co-authored some articles that "may" include questions of causation, but admitted those articles were based on specific patient cases which

"provided the basis of the analysis that took place." Lagana Dep., Dkt. 1718-5, at 117:5-9. The very passage cited by Plaintiffs includes Dr. Lagana's concession that he has never before given a general causation opinion. *Id.* at 119:8-24.

Plaintiffs also offer a list of articles co-authored by Dr. Lagana that reportedly examine the causes of other cancers. But none of these publications are relevant or deal with the supposed etiological link between low-dose nitrosamines and cancer. Plaintiffs do not even address the fact that Dr. Lagana's career in the lab is focused on diagnosing cancerous tissue in slides from cancers that have already manifested in patients. Dr. Lagana has never actually examined or studied a suspected human carcinogen to determine whether in fact it causes cancer and at what dose.

B. Dr. Lagana's Expertise Is Not Relevant to General Causation.

Plaintiffs' Opposition fails to connect Dr. Lagana's expertise in pathology to any of his opinions here – because his expertise is not germane to his opinions. Dr. Lagana's report merely presents conclusions of dietary, occupational, and animal studies and baldly asserts they support Plaintiffs' argument. He provides no specialized lens to assist the jury and no original analysis of those studies. *See Daubert v. Merrell Dow Pharm., Inc.*, 43 F.3d 1311, 1318 (9th Cir. 1995).

Dr. Lagana was criticized in the *Benicar* litigation for failing to apply the Bradford Hill criteria commonly used by epidemiologists to assess causation. Here, he expressly attempted to apply those criteria in order to avoid similar criticism, even

though he conceded in *Benicar* that using the Bradford-Hill Criteria was not a routine part of his practice. Plaintiffs attempt to minimize the concession that his approach was litigation-driven by quoting his testimony that: "We're talking about different things here. . . In this litigation, I haven't looked at any specific cases." (Lagana Dep., Dkt. 1718-5, at 102:8-103:19.) But that is the point: looking at specific cases is the entirety of Dr. Lagana's practice as a pathologist. When asked whether he has ever answered a general causation question in his clinical practice, he admitted "That's not part of my practice." (*Id.* at 107:11-108:14.) In short, while Dr. Lagana may possess certain qualifications as a pathologist and medical doctor, Plaintiffs have not carried their burden of demonstrating that he is qualified to offer the opinions he has rendered, or that he has offered any specialized expertise that would assist the finder of fact in answering the general causation question at issue.

CONCLUSION

Defendants' arguments go beyond the weight to be ascribed to Dr. Lagana's opinions and reach the crux of the type of testimony that Rule 702 and *Daubert* prohibit. Defendants have demonstrated that Dr. Lagana's approach — litigation-driven and deliberately ignorant of critical evidence — is unreliable. For all the foregoing reasons, Defendants respectfully request that the Court exclude or, at minimum, limit Dr. Lagana's opinions.

Dated: January 6, 2022

Respectfully Submitted:

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CERTIFICATE OF SERVICE

I HEREBY CERTIFY that on January 6, 2022, I electronically filed the foregoing with the Clerk of the Court by using the CM/ECF system which will send a notice of electronic filing to all CM/ECF participants in this matter.

/s/ Seth A. Goldberg
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